Walter B. Essman, Ph.D., M.D.
Professor of Psychology and Biochemistry
Queens College of the City University of New York
65-30 Kissena Boulevard
Flushing, New York 11367

Metabolic Response to Stress - Tobacco Smoke Interactions.

This project is directed toward the evaluation of the nature and sequence of metabolic and biochemical changes resulting from stress and/or its systems showing adaptation to stress, and their interaction with the effects produced by nicotine. The basis of the objectives of this work is a definition of those cellular and subcellular units, the biochemical or metabolic composition of which may be altered by either stress, nicotine treatment or the interaction of the two. The indices of stress and the interactive effects thereupon contributed by nicotine treatment will reside within a consideration of biologically active molecules which characterize a cellular or organelle system with which they are classically identified. As such, changes in the content, storage pool levels or turnover of these molecules may well provide extremely sensitive indices of how stress and nicotine treatment interact.

It is not the purpose of this research to explore the multifaceted systems within which stress operates or nicotine treatment exerts measurable effects, but to focus primarily upon five tissue systems from which the cellular and subcellular biochemical data may be derived in terms of those molecules which appear to be most significant for the regulation of physiologic and pathophysiologic processes within such organ systems. Those hypotheses which are generated from the investigator's research findings as well as other related work are: (1) models of acute and chronic stress, respectively, may be developed and may reflect changes that are coincident with the onset, duration and adaptation to stress; (2) several of those changes which are utilized as indices of stress onset, permanence or reversibility may be interacted upon by the effects of nicotine; (3) the nature of the biochemical or metabolic changes observed within representative cellular model systems; and (4) the objective role of nicotine, which serves to either alter the time course for onset of stress indices or change the sequence over which they may occur or be modified will be assessed.

Progress has been significantly made in several areas, notably between stress conditions and the treatment with nicotine and/or tobacco smoke in experimental animals. These findings essentially have implicated nicotine to both the regional and cellular bases in the central nervous system in its interaction with the environmental and physiologic sources of central nervous system stress. Subcellular localization of such effects and the localization of specific synthetic processes with which such interaction is interrelated have been specified.

Activation Date: January 1, 1975

Current Grant Level: $61,254.

1005025527

Source: https://www.industrydocuments.ucsf.edu/docs/zhnk0000